I claim:

1. A method of screening agents to determine if such agents are useful for the prevention and treatment of disorders selected from the group consisting of cachexia and other wasting disorders, cancer, atherosclerosis, heart disease, autoimmune disease, chronic inflammatory disease, alcoholic hepatitis, non-alcoholic hepatitis, rheumatoid arthritis, osteoarthritis, type II diabetes, insulin insensitivity, Parkinson's disease, Alzheimer's disease, and any other condition caused or mediated by chronic oxygen radical damage or by chronic chemical toxicities, said method comprising the steps of:

treating cells of a living organism with a putative agent; and

measuring transient activation of a stress-response pathway,

wherein said transient activation indicates said putative agent is useful for prevention or treatment of said disorders.

2. The method of Claim 1, wherein said stress-response pathway is selected from the group consisting of mitogen activated protein kinase (MAPK) pathways of transcription activation, stress activated protein kinase pathways of transcription activation, hypoxia inducible factor (HIF) pathways of transcription activation, nuclear factor kappa-beta (Nf-κβ) pathways of transcription activation, and insulin-associated GLUT4 activation or translocation pathways.

3. The method of Claim 1, wherein said transient activation includes transient activation of any of the genes or signaling molecules within said pathways.

4. The method of Claim 1, wherein said step of measuring transient activation is performed using a methodology selected from the group consisting of western blot, dot-blot, ELISA, immunocytochemistry, immunohistochemistry, mass-spectroscopy, multiplexed proteomics technology, antibody microarray, or truncated protein analysis to determine enhanced content of gene product(s) in cells, nuclei, or enhanced appearance of gene products within nuclear-protein extracts; phosphorylation-based assays to determine phosphorylation state of any of the gene products, phospholipids, diacylglycerol products or other signaling molecules in said gene or stress-response pathways; gel-shift assays to determine enhanced sequence-specific DNA-binding of the various genes involved in the above-mentioned pathways (such as, but not limited to, AP-1); any reporter-gene assays to determine gene activation; any RT-PCR-based assays (including real-time RT-PCR, in situ hybridization, quantitative and semi-quantitative RT-PCR, and competitive quantitative RT-PCR) to quantify content of mRNA coding for the gene product(s) in question; or any of the RNA- or protein-array or microarray methodologies available to determine gene activation.

5. The method of Claim 1, wherein said transient activation of a stress-response pathway indicates that said putative agent is a source of an agent for treatment of said disorders.

6. The method of Claim 1, wherein said transient activation is longer than approximately 1 minute and is no longer apparent 12 hours after initial activation.

7. The method of Claim 6, wherein said transient activation occurs not more than three times in one day and not less than once each day for three non-consecutive days.

8. The method of Claim 1, wherein said transient activation is produced by a treatment modality selected from the group consisting of modifying dosing regimens, modifying dosing apparatus, modifying dosing formulations, modifying pharmacokinetic properties of said agent, modifying other properties of said agent, modifying vehicle of said agent, and modifying delivery apparatus of said agent.

9. The method of Claim 1 wherein said cells of a living organism are mammalian cells.

10. The method of Claim 9, wherein said mammalian cells are selected from the group consisting of human cells and primate cells.

11. The method of Claim 1, wherein said transient activation of a stress-response pathway is selected from the group consisting of an activation of any of a signaling processes which may result in increased synthesis of any of gene products associated with the stress-response pathways, increased nuclear content of any of gene products associated with the stress-

response pathways, and increased cellular content of any of gene products associated with the stress-response pathways.

12. A method of screening agents to determine if such agents are useful for the prevention or treatment of wasting disorders associated with a disorder selected from the group consisting of cachexia and other wasting disorders, cancer, atherosclerosis, heart disease, autoimmune disease, chronic inflammatory disease, alcoholic hepatitis, non-alcoholic hepatitis, rheumatoid arthritis, osteoarthritis, type II diabetes, insulin insensitivity, Parkinson's disease, Alzheimer's disease, and any other condition caused or mediated by chronic oxygen radical damage or by chronic chemical toxicities, said method comprising the steps of:

treating living organisms with a putative agent; measuring transient activation of a stress-response pathway,

wherein said transient activation indicates said putative agent is useful for prevention or treatment of said wasting disorders.

13. The method of Claim 12, wherein said stress-response pathway is selected from the group consisting of mitogen activated protein kinase (MAPK) pathways of transcription activation, stress activated protein kinase pathways of transcription activation, hypoxia inducible factor (HIF) pathways of transcription activation, nuclear factor kappa-beta (Nf-κβ) pathways of transcription activation, and insulin-associated GLUT4 activation or translocation pathways.

14. The method of Claim 12, wherein said transient activation includes transient activation of any of the genes or signaling molecules within these pathways.

15. The method of Claim 12, wherein said step of measuring transient activation is done using a methodology selected from the group consisting of western blot, dot-blot, ELISA, immunocytochemistry, immunohistochemistry, mass-spectroscopy, multiplexed proteomics technology, antibody microarray, or truncated protein analyses to determine enhanced content of gene product(s) in cells, nuclei, or enhanced appearance of gene products within nuclear-protein extracts; phosphorylation-based assays to determine phosphorylation state of any of the gene products, phospholipids, diacylglycerol products or other signaling molecules in said gene or stress-response pathways; gel-shift assays to determine enhanced sequence-specific DNA-binding of the various genes involved in the above-mentioned pathways (such as, but not limited to, AP-1); any reporter-gene assays to determine gene activation; any RT-PCR-based assays (including real-time RT-PCR, in situ hybridization, quantitative and semi-quantitative RT-PCR, and competitive quantitative RT-PCR) to quantify content of mRNA coding for the gene product(s) in question; or any of the RNA- or protein-array or microarray methodologies available to determine gene activation.

16. The method of Claim 12, wherein said transient activation is longer than approximately 1 minute and is no longer apparent 12 hours after initial activation.

17. The method of Claim 16, wherein said transient activation occurs not more than three times in one day for seven days and not less than once each day for three consecutive days.

18. The method of Claim 12, wherein said transient activation is produced by a treatment modality selected from the group consisting of modifying dosing regimens, modifying dosing apparatus, modifying dosing formulations, modifying pharmacokinetic properties of said agent, modifying other properties of said agent, modifying vehicle of said agent, and modifying delivery apparatus of said agent.

19. The method of Claim 12 wherein said living organisms are mammals.

20. The method of Claim 19, wherein said mammals are selected from the group consisting of humans and primates.

21. The method of Claim 19, wherein said treating living mammals includes treating cultured tissues from said mammals.

22. The method of Claim 21, wherein said cultured tissues are from a human.

23. The method of Claim 12, wherein said transient activation of a stress-response pathway is selected from the group consisting of an activation of any of a signaling processes

which may result in increased synthesis of any of gene products associated with the stress-response pathways, increased nuclear content of any of gene products associated with the stress-response pathways, and increased cellular content of any of gene products associated with the stress-response pathways.